Glycaemia and albuminuria as predictors of coronary heart disease in Aboriginal and Torres Strait Islander adults: a north Queensland cohort

Robyn A McDermott, Brad McCulloch and Ming Li

ABSTRACT

Objective: To evaluate the contribution of non-traditional risk factors to coronary heart disease (CHD) incidence in Indigenous adults.

Design, setting and participants: Cohort study of 1706 Aboriginal and Torres Strait Islander adults from 26 remote communities in far north Queensland who were initially free of CHD, with a mean of 7.5 years of follow-up.

Main outcome measures: CHD-related deaths and hospitalisations obtained by record matching.

Results: CHD incidence was similar in men and women and in Aboriginals and Torres Strait Islanders; overall incidence was 12.1 (95% CI, 10.1–14.1) events per 1000 person-years. At baseline, prevalence of diabetes was 12.4% in Aboriginals and 22.3% in Torres Strait Islanders, prevalence of any albuminuria was similarly high (33.5%) in both groups, and participants with diabetes were 5.5 (95% CI, 4.2–7.3) times more likely to have albuminuria than those without diabetes. At follow-up, adjusted hazard ratios for CHD were 1.7 (95% CI, 1.01–2.8) for obesity based on waist circumference; 1.5 (95% CI, 1.01–2.3) for hypertension; 1.4 (95% CI, 0.9–2.2) for previous or current smoking; 1.9 (95% CI, 1.3–2.7) for elevated triglycerides; 1.3 (95% CI, 0.9–1.9) for low high-density lipoprotein cholesterol; 1.3 (95% CI, 0.8–2.2) for impaired fasting glucose; 2.4 (95% CI, 1.7–3.5) for diabetes; and 4.6 (95% CI, 2.9–7.1) for macroalbuminuria. Baseline albuminuria without diabetes increased risk by 50% (adjusted rate ratio, 1.5 [95% CI, 0.9–2.4]) but diabetes with macroalbuminuria amplified risk sixfold (adjusted rate ratio, 5.9 [95% CI, 3.4–10.1]).

Conclusion: High prevalence of glycaemia and albuminuria in this population, especially when combined, account for much of the excess CHD risk beyond the traditional Framingham risk factors. They can be measured simply, lend themselves to cardioprotective interventions, and should be used routinely to estimate risk and monitor effectiveness of treatment.

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The excess burden of ill health and coronary heart disease (CHD) in Australian Aboriginal and Torres Strait Islander populations is well documented. Rates of hospitalisation for CHD for men and women are 2.4 and 3.9 times, respectively, those for non-Indigenous Australians.[1]

Incidence of CHD in Aboriginal people in the Northern Territory has been reported as more than double that expected, based on the Framingham model, with a particular excess among women.[2] High rates of diabetes account for much of this excess. Similar CHD incidence rates, including the absence of a sex difference, have been reported in an urban Aboriginal population.[3]

Currently, there is significant investment in the implementation of adult health checks for Aboriginal and Torres Strait Islander people. These checks include risk factor assessment that is predominantly based on the Framingham risk factors.[4]

In this study, we aimed to explore the contribution of non-traditional risk factors, especially albuminuria and high fasting glucose, to CHD incidence in a large cohort of Aboriginal and Torres Strait Islander adults from north Queensland.

METHODS

Participants

The Well Person's Health Check (WPHC), conducted between 1998 and 2000, offered screening and referral services for people in 26 remote Indigenous communities in far north Queensland.[5] Of the 2862 Aboriginal and Torres Strait Islander participants aged 15 years and over, 2506 (87.6%) consented to be recontacted for future checks and access to their health records. The study was approved by the Cairns Base Hospital Ethics Committee with support from Apunipima Cape York Health Council and the Torres Strait and Northern Peninsula Area Health Council.

Baseline measurements

Details of the methods used in the WPHC have been published elsewhere.[6] Participants underwent anthropometric assessment, including measurement of waist circumference at the level of the umbilicus. Tobacco smoking and alcohol intake data were collected by a self-report questionnaire. Levels of total cholesterol, high-density lipoprotein cholesterol (HDL-C), triglycerides and glucose were measured from blood collected in the early morning after at least 8 hours of fasting. Blood glucose and lipid levels were measured by photometric enzyme end point assay, using the Cobas Integra 700 and 400 (Roche Diagnostics, New York, NY, USA). Urinary albumin-to-creatinine ratio (UACR) testing was performed routinely by immunoassay on all urine specimens that were collected in a sterile 50 mL container and refrigerated immediately following collection. Sitting blood pressure data were obtained by averaging three measurements taken after 10 minutes rest.

Record matching

Hospitalisation and death records for consenting WPHC participants were identified via a manual search (by a registered nurse with experience working in the region) of the Queensland Health hospital records systems. As there is no unique patient identifier in Queensland, a mapping table, which linked WPHC reference number, hospital facility code and local unit record number, was developed. This table was subsequently applied to the Queensland Hospital Admitted Patient Data Collection, and hospitalisation rele-

1 Baseline characteristics of the study population, 1998–2000*

	Aboriginals		Torres Strait Islanders		Total	
	Women ($n = 534$)	Men ($n = 486$)	Women ($n = 324$)	Men ($n = 362$)	Women ($n = 858$)	Men ($n = 848$)
Age (years)	38.0 (36.7–39.3)	37.2 (35.9–38.6)	39.8 (38.1–41.6)	38.5 (36.9–40.1)	38.7 (37.6–39.7)	37.8 (36.8–38.8)
BMI (kg/m^2)	26.9 (26.2–27.5)	25.0 (24.4–25.5)	33.1 (32.3–33.8)	30.5 (29.9–31.1)	29.2 (28.7–29.7)	27.3 (26.9–27.8)
WC (cm)	94.0 (92.6–95.4)	91.0 (89.6–92.3)	105.6 (103.9–107.4)	102.6 (101.0–104.1)	98.4 (97.3–99.6)	95.9 (94.8–97.0)
Systolic BP (mmHg)	127.0 (125.2–128.8)	132.9 (131.3–134.5)	132.3 (130.0–134.7)	137.8 (136.1–139.5)	129.0 (127.6–130.5)	135.0 (133.8–136.2)
Diastolic BP (mmHg)	70.2 (69.0–71.4)	75.7 (74.4–76.9)	69.0 (67.7–70.4)	74.3 (72.9–75.6)	69.8 (68.9–70.7)	75.1 (74.1–76.0)
Total cholesterol (mmol/L)	4.8 (4.7–4.9)	5.0 (4.9–5.1)	4.9 (4.8–5.0)	5.2 (5.1–5.3)	4.8 (4.7–4.9)	5.1 (5.0–5.2)
Triglycerides (mmol/L)	1.7 (1.6–1.8)	2.1 (1.9–2.3)	1.5 (1.4–1.6)	1.9 (1.7–2.0)	1.6 (1.5–1.7)	2.0 (1.9–2.1)
HDL-C (mmol/L)	1.17 (1.14–1.20)	1.21 (1.17–1.24)	1.10 (1.07–1.13)	1.12 (1.09–1.15)	1.14 (1.12–1.16)	1.17 (1.15–1.19)
Fasting glucose (mmol/L)	5.6 (5.4–5.9)	5.4 (5.2–5.6)	6.3 (5.9–6.6)	6.2 (5.9–6.5)	5.9 (5.7–6.1)	5.8 (5.6–5.9)
UACR	17.3 (11.9–22.8)	13.4 (10.0–16.9)	15.0 (9.4–20.6)	13.9 (9.7–18.0)	16.5 (12.5–20.5)	13.6 (11.0–16.3)
Diabetes, percentage (95% CI)	14.6 (11.6–17.6)	10.3 (7.6–13.0)	24.7 (20.0–29.4)	19.9 (15.8–24.0)	18.4 (15.8–21.1)	14.4 (12.0–16.8)
Smoking (previous and current), percentage (95% CI)	59.2 (55.0–63.4)	71.7 (67.6–75.7)	43.5 (38.1–48.9)	52.1 (46.9–57.2)	53.3 (49.9–56.6)	63.3 (60.0–66.5)

BMI = body mass index. WC = waist circumference. BP = blood pressure. HDL-C = high-density lipoprotein cholesterol. UACR = urinary albumin-to-creatinine ratio.
* Data are mean (SD) unless otherwise specified. ◆

vant to the match unit record, facility code tuples were extracted. Matching of death records was performed manually at the Queensland Registry of Births, Deaths and Marriages.

Outcome determination

The census date for follow-up was 1 January 2006, as this marked the commencement of the mapping of WPHC reference numbers to hospital unit record numbers. Hospitalisations were considered to be CHD related if they contained an International Classification of Diseases, ninth revision, clinical modification (ICD-9-CM) code commencing with 410, 411, 413 or 414, or an ICD-9-CM

2 Incidence of coronary heart disease events per 1000 person-years by age at baseline, sex and ethnicity

	Aboriginals ($n = 1020$)			Torres Strait Islanders ($n = 686$)			Total ($N = 1706$)		
	Events	Person-years	Rate (95% CI)	Events	Person-years	Rate (95% CI)	Events	Person-years	Rate (95% CI)
Women and men									
15–34 years	10	2 889.3	3.5 (1.9–6.4)	4	1 904.7	2.1 (0.8–5.6)	14	4 794.0	2.9 (1.7–4.9)
35–44 years	14	1 387.9	10.1 (6.0–17.0)	3	787.6	3.8 (1.2–11.8)	17	2 175.6	7.8 (4.9–12.6)
45–54 years	24	839.9	28.6 (19.2–42.6)	17	635.4	26.8 (16.6–43.0)	41	1 475.4	27.8 (20.5–37.7)
⩾55 years	28	921.6	30.4 (21.0–44.0)	21	655.5	32.0 (20.9–49.1)	49	1 577.0	31.1 (23.5–41.1)
Total	76	6 038.7	12.6 (10.1–15.7)	45	3 983.3	11.3 (8.4–15.1)	121	10 022.0	12.1 (10.1–14.1)
Women									
15–34 years	6	1 468.4	4.1 (1.8–9.1)	2	904.7	2.2 (0.6–8.8)	8	2 373.1	3.4 (1.7–6.7)
35–44 years	6	712.7	8.4 (3.8–18.7)	2	342.5	5.8 (1.5–23.3)	8	1 055.2	7.6 (3.8–15.2)
45–54 years	12	428.6	28.0 (15.9–49.3)	7	308.7	22.7 (10.8–47.6)	19	737.4	25.8 (16.4–40.4)
⩾55 years	12	523.9	22.9 (13.1–40.3)	10	340.6	29.4 (15.8–54.6)	22	864.5	25.4 (16.8–38.6)
Total	36	3 133.6	11.5 (8.3–15.9)	21	1 896.6	11.1 (7.2–17.0)	57	5 030.1	11.3 (8.7–14.7)
Men									
15–34 years	4	1 420.8	2.8 (1.1–7.5)	2	1 000.1	2.0 (0.5–8.0)	6	2 420.9	2.5 (1.1–5.5)
35–44 years	8	675.3	11.8 (5.9–23.7)	1	445.1	2.2 (0.3–15.9)	9	1 120.4	8.0 (4.1–15.4)
45–54 years	12	411.3	29.2 (16.6–51.4)	10	326.7	30.6 (16.5–56.9)	22	738.0	29.8 (19.6–45.3)
⩾55 years	16	397.7	40.2 (24.6–65.7)	11	314.8	34.9 (19.3–63.1)	27	712.5	37.9 (26.0–55.3)
Total	40	2 905.1	13.8 (10.0–18.8)	24	2 086.7	11.5 (7.7–17.2)	64	4 991.8	12.8 (10.0–16.4)

3 Incidence rate ratios for coronary heart disease (CHD) events by baseline risk factors and ethnicity

	Aboriginals ($n = 1020$)		Torres Strait Islanders ($n = 686$)		Total ($N = 1706$)	
	Crude hazard ratio (95% CI)	Adjusted hazard ratio (95% CI)*	Crude hazard ratio (95% CI)	Adjusted hazard ratio (95% CI)*	Crude hazard ratio (95% CI)	Adjusted hazard ratio (95% CI)*
Body mass index, kg/m^2 (reference category, 18.5–24.99)						
< 18.5	1.4 (0.6–3.3)	1.5 (0.6–3.6)	2.2 (0.3–18.9)	3.1 (0.3–27.1)	1.4 (0.6–3.2)	1.6 (0.7–3.5)
25–29.99	2.1 (1.2–3.9)	1.8 (1.0–3.4)	1.2 (0.4–3.4)	0.7 (0.2–2.0)	1.8 (1.04–2.9)	1.3 (0.8–2.2)
⩾ 30	2.0 (1.1–3.6)	2.1 (1.1–3.8)	1.4 (0.5–3.5)	1.0 (0.4–2.7)	1.7 (1.03–2.7)	1.5 (0.9–2.5)
Waist circumference category† (reference category, normal)						
Overweight	1.4 (0.6–2.9)	1.4 (0.7–2.9)	1.9 (0.5–7.9)	1.2 (0.3–5.2)	1.4 (0.8–2.8)	1.2 (0.6–2.3)
Obese	2.1 (1.2–3.6)	2.1 (1.1–3.8)	3.1 (0.9–9.9)	1.8 (0.5–6.2)	2.1 (1.3–3.4)	1.7 (1.01–2.8)
Reported or measured hypertension (reference category, no)						
Yes	3.6 (2.2–5.9)	2.2 (1.3–3.6)	2.1 (1.1–3.8)	0.8 (0.4–1.6)	2.9 (2.0–4.2)	1.5 (1.01–2.3)
Smoker (reference category, never)						
Previous or current	1.2 (0.7–2.3)	1.4 (0.8–2.6)	1.1 (0.5–2.2)	1.3 (0.6–2.7)	1.2 (0.7–1.9)	1.4 (0.9–2.2)
Urinary albumin-to-creatinine ratio (reference category, < 3.4)						
3.4–33.9	1.7 (1.0–2.9)	1.1 (0.6–1.9)	4.9 (2.3–10.6)	2.4 (1.1–5.5)	2.4 (1.6–3.7)	1.4 (0.9–2.3)
⩾ 34	5.5 (3.2–9.5)	3.2 (1.8–5.7)	13.5 (6.2–29.5)	9.4 (4.3–20.7)	7.4 (4.8–11.3)	4.6 (2.9–7.1)
Fasting glucose quintile, mmol/L‡ (reference category, 1.7–4.4)						
4.5–4.7	0.7 (0.3–1.7)	0.8 (0.3–1.9)	0.8 (0.1–4.6)	0.7 (0.1–4.3)	0.7 (0.3–1.5)	0.7 (0.3–1.6)
4.8–5.1	1.3 (0.6–2.7)	1.0 (0.5–2.1)	1.4 (0.3–6.3)	0.9 (0.2–4.3)	1.3 (0.7–2.5)	0.9 (0.5–1.8)
5.2–5.8	1.2 (0.5–2.6)	0.8 (0.4–1.8)	3.0 (0.8–10.9)	1.6 (0.4–6.1)	1.5 (0.8–2.9)	1.0 (0.5–1.9)
⩾ 5.9	3.0 (1.6–5.7)	1.8 (0.9–3.5)	6.3 (1.9–21.0)	2.6 (0.8–9.1)	3.6 (2.1–6.1)	1.8 (1.04–3.2)
Fasting glucose category, mmol/L (reference category, < 5.5)						
5.5–6.9	2.1 (1.1–3.8)	1.4 (0.7–2.5)	2.6 (1.1–6.4)	1.5 (0.6–3.7)	2.1 (1.3–3.5)	1.3 (0.8–2.2)
⩾ 7.0	3.2 (1.8–5.5)	2.1 (1.2–3.7)	6.1 (3.1–12.0)	3.4 (1.7–6.9)	3.9 (2.6–5.9)	2.4 (1.6–3.6)
Diabetes§ (reference category, no)						
Yes	3.2 (2.0–5.3)	2.3 (1.4–3.8)	4.8 (2.7–8.7)	3.0 (1.6–5.5)	3.7 (2.5–5.3)	2.4 (1.7–3.5)
High-density lipoprotein cholesterol, mmol/L¶ (reference category, > 1.0)						
< 1.0	1.4 (0.8–2.3)	1.2 (0.7–2.0)	1.3 (0.7–2.5)	1.3 (0.7–2.5)	1.4 (0.9–2.0)	1.3 (0.9–1.9)
Triglycerides, mmol/L¶ (reference category, < 2.0)						
⩾ 2.0	2.8 (1.8–4.4)	2.3 (1.5–3.6)	1.5 (0.8–2.8)	1.2 (0.7–2.4)	2.2 (1.5–3.2)	1.9 (1.3–2.7)

* Adjusted for age and sex. † Categories are based on World Health Organization sex-specific criteria. ‡ Analysis includes participants with baseline diabetes. § Defined using World Health Organization criteria. ¶ Categories are based on National Heart Foundation and Australian Diabetes Society recommendations. ◆

procedure code between 3600 and 3699, inclusive. For hospitalisations coded to the International Classification of Diseases, 10th revision, the diagnosis code range I20–I25, and procedure code blocks 669–679, inclusive, were used.

Analysis

Participants who were not considered to be free of CHD at baseline (those who identified a pre-existing heart condition, or had a hospitalisation episode record containing a code for CHD with an admission date before the original screening date) were excluded from the analysis. Those with incomplete baseline clinical data and those reporting non-Indigenous or mixed Indigenous descent (Aboriginal, South Sea Islander and Torres Strait Islander) were also excluded.

Cumulative incidence of CHD events over a mean of 7.5 years was calculated using the Kaplan–Meier method for a range of population strata, including ethnicity, body mass index (BMI), waist circumference category, fasting glycaemia category, presence or absence of diabetes and hypertension, smoking status, fasting triglyceride and HDL-C levels, and urinary albumin-to-creatinine ratio at baseline. Hypertension was defined as blood pressure > 140/90 mmHg. Incidence ratios were calculated using the collapsed, aggregated person-years and events in the strata to be compared, and reported as incidence rate ratios adjusted for age and sex. Further adjustment was made for ethnicity. Hazard ratios were calculated using a Cox proportional hazards model. The model was developed manually and incrementally. All analyses were undertaken using Stata v10.1 (StataCorp, College Station, Tex, USA).

RESULTS

Included in the final analysis were 1706 adults who had complete baseline data and whose records were matched. Of the 2506 WPHC participants who consented to be recontacted, 800 were excluded: 147 were not free of CHD at baseline, 508 had incomplete baseline clinical data, and 145 were of non-Indigenous or mixed Indigenous descent. There was no substantial difference

in age and sex between those excluded and those included. Baseline characteristics of the study population are summarised in Box 1. There were about equal numbers of men and women. The women were slightly older than the men and had a higher prevalence of diabetes. Compared with Aboriginal participants, Torres Strait Islanders were slightly older, had substantially higher BMI, had higher fasting glucose and nearly double the prevalence of diabetes, and had lower smoking rates. Baseline prevalence of diabetes was 12.4% (126/1020) in Aboriginals and 22.3% (153/686) in Torres Strait Islanders, whereas prevalence of any albuminuria (UACR, >3.4) was similarly high (572/1706, 33.5%) in both groups. At baseline, those with diabetes were 5.5 (95% CI, 4.2–7.3) times more likely to have albuminuria than those without diabetes.

One hundred and twenty-one CHD events (deaths and hospitalisations) were recorded in 10 022 person-years of follow-up (Box 2). Incidence of CHD was similar in men and women, and in Aboriginals and Torres Strait Islanders, with an overall incidence rate of 12.1 (95% CI, 10.1–14.1) events per 1000 person-years. Rates increased with age, but there was substantial illness (14 events) in participants aged 15–34 years at baseline.

Because baseline risk categories tracked with age and differed by sex, hazard ratios were adjusted for age and sex (Box 3). Further adjustment for ethnicity did not alter the results. Adjusted hazard ratios for CHD were 1.7 (95% CI, 1.01–2.8) for obesity based on waist circumference; 1.5 (95% CI, 1.01–2.3) for hypertension; 1.4 (95% CI, 0.9–2.2) for previous or current smoking; 1.9 (95% CI, 1.3–2.7) for elevated triglycerides; 1.3 (95% CI, 0.9–1.9) for low HDL-C (<1.0 mmol/L); 1.3 (95% CI, 0.8–2.2) for impaired fasting glucose (5.5–6.9 mmol/L); 2.4 (95% CI, 1.7–3.5) for diabetes; and 4.6 (95% CI, 2.9–7.1) for macroalbuminuria (UACR ⩾ 34). Crude hazard ratio increased with increasing baseline fasting glucose from a threshold of about 5.0 mmol/L but this effect was attenuated after adjustment for age and sex (Box 3).

The Framingham risk factors (BMI, smoking status, blood pressure and hyperlipidaemia) each increased the age-adjusted CHD risk by less than twofold. The stand-out risk predictors for the study population appeared to be albuminuria and glycaemia, which were highly prevalent at baseline in Aboriginal and Torres Strait Islander participants. Compared with those free of both diabetes and albuminuria at baseline, the age- and sex-adjusted risks for diabetes alone and albuminuria alone (Box 4) were similar to the risk based on traditional Framingham risk factors (adjusted rate ratio, 1.4 [95% CI, 0.9–2.4]). However, diabetes coexisting with microalbuminuria and macroalbuminuria amplified the adjusted rate ratio to 2.3 (95% CI, 1.3–4.2) and 5.9 (95% CI, 3.4–10.1), respectively (Box 4).

DISCUSSION

In our study, the adult population of Indigenous Australians from north Queensland had a very high background prevalence of diabetes and albuminuria and an excess incidence of CHD compared with the non-Indigenous population. Age-specific CHD incidence rates were similar for men and women, and for Aboriginals and Torres Strait Islanders, and comparable to those reported for Aboriginal adults in the Northern Territory and Western Australia.[2,3] The higher prevalence of diabetes among women that we observed could explain the lack of sex "protection", and follows patterns described in other populations where the stronger effect of diabetes in females can be partly explained by a heavier risk factor burden and greater effect of blood pressure and atherogenic dyslipidaemia.[7,8] The Strong Heart Study found a similar increased risk of CHD in Native American tribes with similarly high baseline levels of diabetes and albuminuria, and developed a "risk calculator" that takes these factors into account.[9]

Our study was limited by possible under-identification of matching hospitalisation and death records, as searching was done manually and deaths are often registered late. Also, there was potential for miscoding of hospital separation data and cause of death in routine collections. However, as most of the communities included were small, we could verify most deaths, including cause of death, from local clinical records and staff.

Our finding that a glycaemia threshold lower than that for a diagnosis of diabetes also increases CHD risk in the study population accords with data from other, larger studies. A meta-analysis of 38 prospective studies found a pooled risk ratio of 1.26 (95% CI, 1.11–1.43) for cardiovascular disease end points among non-diabetic subjects with elevated blood glucose regardless of the type of blood glucose assessment.[10] The risk was higher in cohorts including women than in cohorts of men, and the analysis suggested a threshold effect of fasting glucose of 5.6 mmol/L, which supports the revised criteria for the diagnosis of impaired fasting glucose by the American Diabetes Association.[11] A pooled analysis of 17 Asian and Australian cohorts found a threshold of 4.9 mmol/L; overall, there was a 23% lower risk of ischaemic heart disease for each 1 mmol/L lower usual fasting glucose level.[12] These analyses suggest that the pathogenic role of hyperglycaemia on blood

4 Coronary heart disease (CHD) events, incidence rates and rate ratios by baseline diabetes and albuminuria* categories

	No.	Person-years	Events	Incidence rate (95% CI)	Crude rate ratio (95% CI)	Adjusted rate ratio† (95% CI)
No diabetes or albuminuria	1045	6244.9	39	6.2 (4.6–8.5)	Reference	
Diabetes only	89	534.0	5	9.4 (3.9–22.5)	1.5 (0.6–3.9)	1.0 (0.4–2.6)
Albuminuria only	381	2230.6	34	15.2 (10.9–21.3)	2.5 (1.5–3.9)	1.5 (0.9–2.4)
Either diabetes or albuminuria	470	2764.7	39	14.1 (10.3–19.3)	2.3 (1.5–3.6)	1.4 (0.9–2.2)
Diabetes and microalbuminuria	121	671.1	19	28.3 (18.1–44.4)	4.6 (2.7–8.0)	2.3 (1.3–4.2)
Diabetes and any albuminuria	191	1012.4	43	42.5 (31.5–57.3)	6.9 (4.5–10.6)	3.6 (2.4–6.0)
Diabetes and macroalbuminuria	70	341.3	24	70.3 (47.1–104.9)	11.2 (6.7–18.6)	5.9 (3.4–10.1)

UACR = urinary albumin-to-creatinine ratio. * Albuminuria, UACR ⩾ 3.4; microalbuminuria, UACR ⩾ 3.4 to < 34; macroalbuminuria, UACR ⩾ 34. † Adjusted for age and sex; further adjustment for ethnicity did not change rate ratios. ◆

vessel walls exists in the early stages of glucose intolerance. The effect of postprandial glucose levels in these larger studies seems to be greater than that of fasting glucose, so our estimates may be conservative. The most appropriate targets in management may, therefore, be postprandial glucose or glycated haemoglobin levels.[13]

In patients with diabetes, increasing albuminuria is associated with marked increases in risk of CHD and all-cause mortality, and reduction in albuminuria during treatment translates to reduction in cardiovascular events.[14,15] Similarly, in non-diabetic subjects (with or without elevated glycaemia), reduction in albuminuria translates to reduction in cardiovascular end points.[16]

The great amplification of CHD risk when impaired fasting glycaemia or diabetes and albuminuria coexist, which we found in our study population, highlights the need for attention to both glycaemia and albuminuria in management plans. High fasting glucose levels and albuminuria found during routine adult health checks should, therefore, be actively managed and monitored, even in the absence of overt diabetes. Reduction in glycaemia and albuminuria should be basic therapeutic goals for CHD risk reduction in high-risk populations such as Indigenous Australians; measures of glycaemia and albuminuria should be routinely reported, in addition to the traditional measures of blood pressure, lipid levels, tobacco smoking and weight.

The challenge we now face is to apply this new knowledge about easy-to-measure clinical predictors of excess CHD incidence in clinical settings, with particular attention to our highest-risk patients — those who have poor access to healthy food, health care services, and other infrastructure and life choices. This includes attending to the needs of the whole person (who may have several serious comorbidities) in a family and community context.

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COMPETING INTERESTS

None identified.

AUTHOR DETAILS

Robyn A McDermott, FAFPHM, PhD, Professor of Public Health[1]
Brad McCulloch, BSc, MPH, Manager[2]
Ming Li, MD, PhD, Research Fellow[1]
1 Sansom Institute for Health Research, University of South Australia, Adelaide, SA.
2 Tropical Public Health Unit, Queensland Health, Cairns, QLD.
Correspondence:
Robyn.Mcdermott@unisa.edu.au

REFERENCES

1 Australian Institute of Health and Welfare. The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples. Canberra: AIHW, 2008. (AIHW Cat. No. IHW 21.)
2 Wang Z, Hoy W. Association between diabetes and coronary heart disease in Aboriginal people: are women disadvantaged? *Med J Aust* 2004; 180: 508-511.
3 Bradshaw PJ, Alfonso HS, Finn JC, et al. Coronary heart disease events in Aboriginal Australians: incidence in an urban population. *Med J Aust* 2009; 190: 583-586.
4 Queensland Health, Royal Flying Doctor Service, editors. Chronic disease guidelines. 2nd ed. Cairns: Queensland Health, 2007.
5 Miller G, McDermott R, McCulloch B, et al. The Well Person's Health Check: a population screening program in indigenous communities in north Queensland. *Aust Health Rev* 2002; 25: 136-147.
6 McDermott RA, Li M, Campbell SK. Incidence of type 2 diabetes in two Indigenous Australian populations: a 6-year follow-up study. *Med J Aust* 2010; 192: 562-565.
7 Juutilainen A, Kortelainen S, Lehto S, et al. Gender difference in the impact of type 2 diabetes on coronary heart disease risk. *Diabetes Care* 2004; 27: 2898-2904.
8 Huxley R, Barzi F, Woodward M. Excess risk of fatal coronary heart disease associated with diabetes in men and women: meta-analysis of 37 prospective cohort studies. *BMJ* 2006; 332: 73-77.
9 Lee ET, Howard BV, Wang W, et al. Prediction of coronary heart disease in a population with high prevalence of diabetes and albuminuria: the Strong Heart Study. *Circulation* 2006; 113: 2897-2905.
10 Levitan EB, Song Y, Ford ES, Liu S. Is nondiabetic hyperglycemia a risk factor for cardiovascular disease? A meta-analysis of prospective studies. *Arch Intern Med* 2004; 164: 2147-2155.
11 American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care* 2004; 27 Suppl 1: S5-S10.
12 Lawes CM, Parag V, Bennett DA, et al; Asia Pacific Cohort Studies Collaborative. Blood glucose and risk of cardiovascular disease in the Asia Pacific region. *Diabetes Care* 2004; 27: 2836-2842.
13 Milicevic Z, Raz I, Beattie SD, et al. Natural history of cardiovascular disease in patients with diabetes: role of hyperglycemia. *Diabetes Care* 2008; 31 (Suppl): S155-S160.
14 Nag S, Bilous R, Kelly W, et al. All-cause and cardiovascular mortality in diabetic subjects increases significantly with reduced estimated glomerular filtration rate (eGFR): 10 years' data from the South Tees Diabetes Mortality study. *Diabetic Medicine* 2007; 24: 10-17.
15 de Zeeuw D, Remuzzi G, Parving H-H, et al. Albuminuria, a therapeutic target for cardiovascular protection in type 2 diabetic patients with nephropathy. *Circulation* 2004; 110: 921-927.
16 Ibsen H, Olsen MH, Wachtell K, et al. Reduction in albuminuria translates to reduction in cardiovascular events in hypertensive patients: losartan intervention for endpoint reduction in hypertension study. *Hypertension* 2005; 45: 198-202.

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